

Environmental lead exposure, Catechol-O-methyltransferase (COMT) Gene, and Systolic Blood Pressure in Children.

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Objective: Environmental lead exposure and a COMT polymorphic variant, Val108/158Met (rs4680), have both been shown to be associated with increased risks of hypertension in adults. We hypothesized that low-level lead exposure influences blood pressure in children and that the COMT mutation modifies this effect. We tested these hypotheses in 309 children at 7–15 years of age from a sub-study of the Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) project, an environmental birth cohort study.

Materials and Methods:

We used a mercury sphygmomanometer with appropriate-size cuffs to measure systolic and diastolic blood pressure (SBP and DBP), and inductively coupled plasma mass spectrometry (ICPMS) to measure blood lead levels (BLLs); all measurements were taken concurrently. Multiple linear regression models were used to estimate the association between blood pressure and BLL, and effect modification by COMT genotype.

Results: Of the 309 children, 126 (42.4%), 136 (45.8%) and 35 (11.8%) were COMT Val/Val, Val/Met and Met/Met genotype, respectively. The median [interquartile-range (IQR)] concurrent BLL was 3.4 (1.83) µg/dl. After adjusting for child's age, height, body mass index, gestational age, weight at birth, and maternal education blood pressure, smoking during pregnancy, an interquartile range increase in BLL (1.83 µg/dl) was associated with 2.98 mm Hg (95% CI 0.42-5.54 mm Hg) increase in SBP among Val/Val wild-type children, while no significant effects were found for DBP. Compared with children having Val/Met or Met/Met genotype, BLL was significantly associated with steeper increases in SBP in children having Val/Val wild-type (p for interaction = 0.02).

Conclusion: These results indicate that concurrent low environmental lead exposure (less than 10 µg/dl) was associated with increased SBP in children, and that genetic polymorphisms in COMT may modify lead's toxicity, suggesting a gene-environment interaction.